We claim:

1. A method of preventing or treating IgA mediated autoimmune disorders, the method comprising:

identifying a subject having an IgA mediated autoimmune disorder; and

providing to the subject a therapeutically effective amount of an agent selected from the group consisting of uteroglobin, or a fragment, derivative, mimetic, or other variant thereof, which prevents or improves the IgA mediated autoimmune disorder.

2. The method of claim 1, wherein the autoimmune disorder is selected from the group consisting of IgA nephropathy, Wegener's granulomatosus, Goodpasture's disease, or diabetic glomerulosclerosis.

3. The method of claim 2, wherein the autoimmune disorder is IgA nephropathy.

4. The method of claim 1, wherein providing the agent comprises administering uteroglobin, or a therapeutically effective variant thereof.

5. The method of claim 4, wherein the administering comprises administering uteroglobin.

6. The method of claim 4, wherein the therapeutically effective variant comprises a polypeptide having at least 85% homology to uteroglobin.

7. The method of claim 6, wherein the therapeutically effective variant comprises a polypeptide having at least 95% homology to uteroglobin.

8. The method of claim 1, wherein providing the agent comprises stimulating endogenous production of uteroglobin in the subject.

9. A method of screening for a derivative, mimetic or variant of uteroglobin that prevents or treats an IgA mediated autoimmune disorder, comprising:

providing a recombinant, non-human mammal having cells that normally express uteroglobin, wherein the cells have been altered to reduce or

eliminate expression of uteroglobin, and predispose the mammal to develop the IgA mediated autoimmune disorder;

administering to the mammal a test agent, to determine if the test agent interferes with development of the IgA mediated autoimmune disorder; and

detecting the presence or absence of the IgA mediated autoimmune disorder in the mammal.

10. The method of claim 9, wherein the IgA mediated autoimmune disorder is IgA nephropathy.

11. The method of claim 9, wherein the cells of the mammal contain a pair of uteroglobin alleles, and the cells are altered by disrupting both alleles so that they do not express endogenous uteroglobin.

12. The method of claim 4, wherein both alleles are disrupted by insertion of a foreign nucleic acid sequence in a DNA sequence of each allele.

13. The method of claim 9, wherein the cells are altered by expression of an antisense nucleotide that reduces or eliminates expression of uteroglobin.

14. A method of screening for an agent that prevents or treats IgA nephropathy, comprising:

administering a test agent to the recombinant mammal of claim 9; and

determining whether the mammal develops IgA nephropathy.

15. The method of claim 9, wherein the test agent is a fragment, derivative, mimetic, or variant of uteroglobin, which prevents or improves the IgA mediated autoimmune disorder.

16. A method of screening for an agent that prevents or treats an IgA mediated autoimmune disease, the method comprising:

providing a cell or cellular extract that expresses a functional uteroglobin receptor;

contacting a sufficient amount of a test compound with the cell or cellular extract to determine whether the test compound binds to the receptor with high affinity; and

selecting the test agent for further testing if it binds to the receptor with high affinity.

17. The method of claim 16, wherein the further testing comprises the method of claim 9.

18. The method of claim 1, further comprising administering a second therapeutic agent to the subject, wherein the second therapeutic agent is effective in treating or preventing the IgA mediated autoimmune disorder.

19. The method of claim 1, wherein the second therapeutic agent is a corticosteroid.

20. The method of claim 1, wherein administering comprises administering recombinant uteroglobin, or a recombinant fragment or variant thereof.

21. The method of claim 1, wherein the therapeutically effective amount of uteroglobin, or a fragment, derivative, mimetic, or variant thereof, is administered by an endotracheal, pulmonary inhalation, ophthalmic, intravenous, intraperitoneal, intramuscular, subcutaneous, transdermal, intradermal, intracranial ventricular, intrathecal, or oral route.

22. The method of claim 1, wherein the therapeutically effective amount of uteroglobin, or a fragment, derivative, mimetic, or variant thereof, has a purity of greater than about 75%.

23. The method of claim 22, wherein the purity is greater than about 95%.

24. A method of predicting susceptibility to IgA nephropathy in a subject, comprising measuring a level of uteroglobin in a biological material from the subject, and determining if the uteroglobin level is below a normal level.

25. The method of claim 24, wherein the biological material is blood.

26. The method of claim 24, wherein the subject is suspected of having an IgA nephropathy, and a level of uteroglobin below the normal level indicates a diagnosis of IgA nephropathy.

27. A method of diagnosing IgA nephropathy, comprising determining whether a subject has an abnormally low level of uteroglobin in a biological material from the subject.

28. The method of claim 27, wherein the biological material is blood.

29. The method of claim 27, wherein the biological material is urine.

30. A method of detecting a predisposition to developing asthma or an IgA mediated autoimmune disorder in a subject, comprising:

obtaining a sample of nucleic acid from the subject;

screening for a polymorphism selected from the group consisting of: (a) an A-to-G polymorphism at position 38 in exon 1 of the uteroglobin gene; and (b) a polymorphism comprising a variation in a number of (GTTT) repeats between about bp -3200 and -3100.

31. The method of claim 30, wherein the method is a method of detecting a predisposition to develop asthma.

32. The method of claim 30, wherein the method is a method of detecting a predisposition to develop an IgA mediated autoimmune disorder.

33. The method of claim 30 wherein the method comprises screening for an A-to-G polymorphism at position 38 in exon 1 of the uteroglobin gene.

34. The method of claim 30 wherein the method comprises screening for a polymorphism comprising variation in the number of (GTTT) repeats between about bp -3200 and -3100.

35. The method of claim 32, wherein the IgA mediated autoimmune disorder is IgA nephropathy.

36. The method of claim 1, wherein the IgA mediated autoimmune disorder is a pulmonary disease.

37. The method of claim 36, wherein the pulmonary disease is a pulmonary inflammatory disease.

38. A method of treating pulmonary inflammation in a subject, comprising administering to the subject a therapeutically effective amount of an agent selected from the group consisting of uteroglobin, or a fragment, derivative, mimetic, or other variant thereof, which prevents or improves the pulmonary inflammation.

39. A composition for use in inhibiting or treating an IgA mediated disorder, comprising a therapeutically effective amount of an agent selected from

the group consisting of uteroglobin, or a fragment, derivative, mimetic, or other variant thereof, which prevents or improves the pulmonary inflammation.

40. The composition of claim 39, wherein the autoimmune disorder is selected from the group consisting of IgA nephropathy, Wegener's granulomatosus, Goodpasture's disease, or diabetic glomerulosclerosis.

41. The composition of claim 40, wherein the autoimmune disorder is IgA nephropathy.

42. The composition of claim 39, wherein the agent comprises uteroglobin, or a therapeutically effective variant thereof.

43. The composition of claim 42, wherein the composition comprises uteroglobin.

44. The composition of claim 42, wherein the therapeutically effective variant comprises a polypeptide having at least 85% homology to uteroglobin.

45. The composition of claim 44, wherein the therapeutically effective variant comprises a polypeptide having at least 95% homology to uteroglobin.

46. The composition of claim 1, wherein the agent comprises an agent that stimulates endogenous production of uteroglobin in the subject.